INTRODUCTION

Obesity produces measurable reductions in pulmonary function and is strongly associated with breathing disorders in sleep, such as sleep apnoea and obesity–hypoventilation.

Moderate to severe degrees of obesity can lead to a restrictive abnormality in lung function due to the mechanical effects of central body fat. Similar fat deposition is linked to upper airway collapsibility in sleep and recent epidemiological data have identified obesity as a crucial risk factor in the development of obstructive sleep apnoea (OSA). Moreover, the combination of obesity-reduced pulmonary function and sleep apnoea can lead to progressive respiratory failure in sleep finally resulting in awake respiratory failure (obesity–hypoventilation syndrome).

Sleep-disordered breathing has a number of clinical consequences, including excess cardiovascular morbidity. Obesity is an important confounder of this association. Conservative measures such as weight reduction may reduce apnoea severity but long-term maintenance of weight reduction is a limiting factor. Treatment of sleep-breathing disorders has been advanced greatly by the use of positive airway pressure devices.

PULMONARY FUNCTION IN OBESITY

Pulmonary Function and Mechanics

Fat deposition in the neck, upper airway, chest wall and abdomen can impair the mechanical function of the respiratory system. In general, the effects of obesity alone are mild and are typically in proportion to the degree of obesity (1–3). Reduced lung volumes are seen, with falls in the expiratory reserve volume (ERV) and the functional residual capacity (FRC) the commonest findings. Reductions in vital capacity and total lung capacity are generally only seen when the body mass index (BMI) exceeds 40 kg/m². Reductions in lung volumes below 70% predicted are rarely due to obesity alone. Measurements of central obesity may correlate more closely than BMI with abnormalities of lung function (4,5). Patients with obesity–hypoventilation syndrome (OHS) tend to have more impaired respiratory function than patients without sleep-disordered breathing, despite identical degrees of obesity. The reasons for this are not clear.

In obese subjects, both airway and respiratory resistance are higher than normal and increase as BMI increases (2). The reduced lung volumes of obesity, in particular the low FRC, explain a large part of the increased resistance. Respiratory resis-
tance increases further when obese subjects are supine (6). Despite this increased resistance, the \( FEV_1/FVC \) (forced expiratory volume in 1 second/forced vital capacity) ratio is normal (1,2,7–9). Studies on total compliance of the respiratory system in obese subjects have found conflicting results, but lung compliance is probably reduced by around 25% (7). The reasons for this are unclear, but small airway closure and collapse may be responsible.

The fraction of total oxygen consumption dedicated to respiratory muscle work during quiet breathing can be up to 15% (i.e. five times normal) in patients with morbid obesity (10), suggesting significantly increased work of breathing. Respiratory muscle function, measured by maximal inspiratory and expiratory pressures, is normal in eucapnic obese subjects. In OHS, inspiratory muscles are weaker, possibly due to the effects of hypoxaemia and hypercapnia. There is little evidence of morphometric differences in respiratory muscles in obesity.

**Gas Exchange**

Hypoxaemia, with or without hypercapnia, is seen in many patients with morbid obesity but many patients with similar degrees of obesity have normal gas exchange (11–13). In obese hypoxaemic subjects, ventilation–perfusion mismatching has been demonstrated, with dependent well-perfused areas of lung relatively underventilated, probably due to partial airway collapse (14,15). The gas exchange abnormalities are usually greater when patients are supine.

More recently, the presence of sleep-disordered breathing has been found to impact significantly on gas exchange abnormalities and may explain some of the variation found in obese subjects. Obesity–hyperventilation syndrome (OHS) describes hypercapnic hypoxaemic respiratory failure in obese patients who have no significant lung disease. The respiratory failure is largely due to impaired ventilatory control secondary to sleep-disordered breathing, resulting in chronic awake alveolar hypoventilation. Patients with OHS can attain a normal \( P_{aCO_2} \) by voluntary hyperventilation (16). However the A–a\( P_{O_2} \) (alveolar–arterial oxygen gradient) is often increased in patients with OHS, suggesting the presence of increased ventilation–perfusion mismatch in addition to hypoventilation. OSA without OHS can also contribute to awake gas exchange abnormalities. Laaban et al. (17) studied a group of 60 obese subjects (BMI around 50kg/m\(^2\)) and found that daytime hypoxaemia was significantly correlated with the presence of OSA. Similar findings have been reported by Gold et al. (13). This mild to moderate hypoxaemia with eucapnia seen in obese patients with OSA is probably attributable to abnormalities of ventilatory control causing mild hypoventilation: this group may represent an early form of OHS.

Interestingly, the single-breath diffusing capacity for carbon monoxide (DLCO), a measure of gas exchange capacity of the lung, is increased in obese subjects by about 10%, with increases in KCO (DLCO adjusted for lung volume) of around 25% (18). The cause is unclear, but may be related to increased pulmonary capillary blood volume.

**Ventilatory Control**

Assessment of ventilatory control, usually measured by responses to chemical stimuli such as hypercapnia and hypoxaemia, is complicated by the wide variation in normal responses. In most patients with simple obesity, ventilatory drive is normal. Reduced ventilatory responses to hypercapnia have been reported in patients with OSA (13). Patients with OHS often have blunted ventilatory responses to hypoxia and hypercapnia though typically there is a shift in CO\(_2\) responsiveness, characterized by a normal slope of the ventilatory response to CO\(_2\), albeit at a higher level of \( P_{aCO_2} \) (19). However, other findings have been mixed. The ability of patients with OHS to voluntarily hyperventilate their \( P_{aCO_2} \) to normal levels implies impaired control. Both familial factors and lifetime alcohol intake can influence ventilatory drive.

**Respiratory Symptoms and Obesity**

Some studies have reported an increased incidence of respiratory symptoms, in particular exertional breathlessness and chest discomfort in obese, otherwise healthy subjects (20,21). Interestingly when comparing subjects with similar degrees of obesity, those patients with OSA report more exertional
dyspnoea than those without OSA (22). Weight loss following bariatric surgery results in significant relief of these symptoms (23), with an independent association between the reduction in sleep-disordered breathing and relief of breathlessness and chest pain. This suggests that OSA is implicated in the genesis of these symptoms in subjects with obesity, possibly through effects on respiratory control with relative daytime hypoventilation and resulting mild hypoxaemia. In the absence of weight loss, continuous positive airway pressure (CPAP) therapy can improve daytime gas exchange and respiratory control (24,25) in patients with OSA and so may also reduce the incidence of daytime respiratory symptoms in obese subjects with OSA.

There have been a number of recent studies describing an epidemiological association between obesity and asthma (26,27). However, the diagnosis of asthma in these studies was not confirmed with tests of bronchial hyperresponsiveness (BHR). A more recent study (28) showed an increased incidence of doctor-diagnosed asthma and asthma medication usage in obese subjects but found no increase in the incidence of BHR or atopy in obese subjects compared to normals. This suggests that asthma is over-diagnosed and over-treated in obese subjects, probably due to the respiratory symptoms associated with obesity alone. Weight loss, either by dietary means or by bariatric surgery, can result in a reduction in asthma symptoms and medication usage (29,30). However, these studies have not demonstrated changes in BHR. This suggests that the reduction in respiratory symptoms is due to the reduction in weight and improvement in obesity-associated conditions such as sleep-disordered breathing or gastro-oesophageal reflux, rather than to a change in the severity of asthma.

**What is Sleep Apnoea?**

Sleep-disordered breathing encompasses a spectrum of conditions ranging from snoring through to profound nocturnal hypoventilation and respiratory failure. Obstructive sleep apnoea (OSA) is characterized by repetitive cessation of airflow during sleep (apnoea), secondary to collapse of the upper airway at the level of the pharynx (31) (Figure 27.1). During apnoeas, respiratory efforts continue against the closed airway and hypoxaemia occurs, until the apnoea is terminated by arousal and upper airway patency is re-established. In the typical patient, after a few deep breaths (often loud snores), the cycle of events is repeated as often as 200–600 times per night. As a result of recurrent arousals, sleep is dramatically fragmented with loss of normal sleep architecture. This, in turn, results in loss of vigilance or even severe sleepiness during the day (32).

Clinically significant upper airway obstruction may occur in the absence of complete collapse of the upper airway. Partial obstruction (hypopnoea) may produce similar pathophysiological events (i.e. hypoxaemia and arousal). Even minor increases in airway resistance can be associated with repetitive arousal and excessive daytime sleepiness, the ‘upper airway resistance’ syndrome (33).

At the other end of the spectrum are patients with severe respiratory failure in sleep including patients with chronic lung disease, respiratory muscle failure due to neuromuscular disorders and the obesity–hypoventilation syndrome (OHS). These patients have prolonged periods of hypoxaemia usually due to reduced ventilation (lasting minutes) rather than apnoeas (lasting 10–60 seconds typically). The hypoventilation causes progressive hypercapnia in sleep, leading to resetting of central chemoreceptors and tolerance of higher awake carbon dioxide tensions. The prolonged exposure to hypoxaemia and hypercapnia can cause or worsen pulmonary hyper-
Pathogenesis of Sleep Apnoea

Collapse of the upper airway occurs when the negative (or suction) pressure applied to the upper airway during inspiration is greater than the dilating force applied by upper airway muscles, such as genioglossus (31,32). Any factors which reduce airway size, decrease muscle tone, increase upper airway compliance or lead to generation of a greater inspiratory pressure will predispose to OSA. Muscle tone and suction pressure are influenced by sleep stage and relative respiratory drive to the diaphragm versus the upper airway dilator muscles.

In general, obese sleep apnoea patients have larger tongues and smaller upper airway volumes than normal subjects (35). However, excess fat deposition around the airway is not a universal finding in obese OSA patients (36) and well-matched controls are often difficult to obtain. Neck fat deposition promotes mass loading and obstruction of the upper airway in sleep, leading to OSA (37) and in morbidly obese patients, neck size is a better predictor of sleep apnoea than other body anthropomorphic measures (22). In addition, abdominal obesity may reduce lung volumes and reflexly reduce pharyngeal cross-sectional area and increase pharyngeal resistance (38,39). Obesity may promote sleep apnoea through multiple mechanisms: in some patients, subcutaneous neck fat may be the critical factor causing upper airway closure in sleep; in other patients, abdominal fat loading may be important.

Obesity–Hypoventilation Syndrome

When awake, the majority of patients with sleep apnoea have normal arterial carbon dioxide tensions. The original descriptions of OSA emphasized the minority of patients with awake respiratory failure who were labelled ‘Pickwickian syndrome’ (see Kryger (40) for review). The recognition that sleep apnoea was present in these patients and that relief of upper airway obstruction by tracheostomy effectively treated the respiratory failure altered the understanding of the evolution of OHS. Upper airway obstruction is clearly a crucial factor in the pathogenesis of OHS (34). However, since most OSA patients do not have hypercapnia when awake, upper airway obstruction alone is insufficient to cause OHS. Similarly, obesity is not a pre-requisite to develop respiratory failure in OSA and obesity, per se, is associated with normal chemosensitivity. A number of recent studies have emphasized the multifactorial aetiology of awake respiratory failure in OSA. The key elements are a combination of obesity (increased upper airway loading and reduced lung volumes), airflow limitation, poor chemoreceptor function (particularly defective arousal responses to hypoxia) and possibly alcohol consumption (reducing upper airway tone tension and right-sided heart failure, ‘cor pulmonale’).
and arousal responses to asphyxia) (34). It is important to stress that awake hypercapnia can occur in obese patients in the absence of any smoking history or lung disease (9).

Longitudinal studies demonstrating the development of OHS are lacking but almost certainly the severity of sleep-induced respiratory abnormalities is crucial in the development of OHS (9). During an apnoea, $P_{\text{aCO}_2}$ rises and $P_{\text{aO}_2}$ falls. When the apnoea is terminated by an arousal, ventilation increases and oxygen and carbon dioxide levels can return to normal. If arousal responses or ventilatory responses to either hypoxia or hypercapnia are depressed, the apnoeic periods will be longer, the degree of blood gas derangement greater and normalization of blood gases in the period following arousal compromised (34). In those patients able to increase ventilation between apnoeas, overall eucaopia will be maintained. In contrast, if the compensatory mechanisms are poor, ventilation will be inadequate during sleep. This will eventually allow the resetting of the chemoreceptors (19) and progression to daytime $CO_2$ retention. Arousal responses may further be impaired in patients prescribed sedatives/hypnotics to improve ‘insomnia’, opiate analgesics to ease musculoskeletal pain or by consumption of alcohol. This ‘vicious cycle’ will progress over time to cor pulmonale if left untreated. Alternatively there is a risk of sudden death due to an arrhythmia in sleep (41).

### SLEEP-DISORDERED BREATHING—CLINICAL

#### Symptoms of Sleep-disordered Breathing

The dominant symptoms associated with OSA are heavy snoring and excessive daytime sleepiness (EDS). Witnessed apnoeas may be a relatively specific symptom in patients but is relatively insensitive. Other symptoms are listed in Table 27.1. Daytime symptoms include morning headaches, fatigue, poor memory and concentration, alteration in mood and impotence (32).

The nature of these symptoms emphasizes the importance of obtaining a history from the spouse, bed partner and other family members. Few patients are aware that they snore or stop breathing during sleep. Excessive sleepiness may be recognized by the patient, but often is either denied by the patient or considered to be ‘normal’—again underlining the critical importance of confirmatory history from a family member, friend or workmate.

Examination of the upper airway may be important. The uvula and soft palate are often swollen and oedematous in patients with sleep apnoea due to the vibration of soft tissues with snoring.

<table>
<thead>
<tr>
<th>Table 27.1 Symptoms of sleep-disordered breathing</th>
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<tbody>
<tr>
<td>Snoring</td>
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<td>Choking in sleep</td>
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<td>Disrupted sleep at night</td>
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<td>Daytime sleepiness</td>
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<td>Dry throat</td>
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<td>Palpitations in sleep</td>
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<td>Nocturia</td>
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<td>Heartburn</td>
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<td>Headaches (day or night)</td>
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<td>Fatigue</td>
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<tr>
<td>Poor memory and concentration</td>
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<tr>
<td>Alteration in mood, irritability</td>
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<td>Impotence</td>
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#### Clinical Sequelae

**PsychoSocial**

Excessive daytime sleepiness (EDS) is characteristic but not pathognomonic of sleep apnoea. Sleepiness in OSA is predominantly related to the repetitive arousals and sleep fragmentation, but a direct effect of hypoxaemia is possible (42). There is a relatively poor correlation between severity of OSA and daytime sleepiness and no simple test accurately quantifies daytime sleepiness. Sleepiness may lead to both impaired work performance and impaired driving (43,44). Some studies have suggested sleep apnoea is a significant risk in commercial drivers. Treatment with nasal CPAP dramatically improves daytime sleepiness, quality of life and even driving simulator performance. A number of studies have found OSA patients perform poorly on psychometric tests compared to controls with a variable degree of improvement following nasal CPAP therapy (42,45). Data from the Swedish Obese Subjects (SOS) Study indicates that in equally obese men and women, a history of sleep apnoea is associated with impaired work performance, increased sick
leave and a much higher divorce rate (46).

**Cardiovascular Sequelae of Sleep Apnoea**

Patients with sleep apnoea clearly have acute cardiovascular changes as an immediate consequence of their breathing disturbance. Obstructive apnoeas are accompanied by profound haemodynamic changes with increases in both systemic and pulmonary arterial blood pressure. With progressive apnoea, there is worsening hypoxaemia, increasing pleural pressure swings, bradycardia (and possibly bradyarrhythmias), increased sympathetic nerve activity and an overall rise in blood pressure. These marked changes in cardiorespiratory behaviour, together with reported changes in cerebral blood flow, provide an environment for increasing the risk of various vascular disease endpoints. Studies using a canine model of OSA have shown that sustained hypertension develops after 1–3 months of OSA (47). Similarly, studies with rats have found that intermittent hypoxia induces a persistent increase in diurnal blood pressure, possibly mediated through renal sympathetic nerve activity and the renin–angiotensin system (48).

Sleep apnoea is a common finding in hypertension clinic patients, but there are confounding factors such as central obesity and increasing age (49,50). A number of studies have strongly suggested that sleep apnoea is a risk factor for hypertension independent of obesity (22,50–53). Recently published cross-sectional data (54) from the Sleep Heart Health Study found a significant association between sleep-disordered breathing and hypertension after adjustment for BMI, neck circumference and waist-to-hip ratio. Similarly, prospective data from the Wisconsin Sleep Cohort Study (55) found a dose–response association between sleep-disordered breathing and hypertension, independent of measures of obesity. Studies using either intrarterial monitoring, automated daytime blood pressure readings or 24-hour ambulatory blood pressure have demonstrated a fall in blood pressure levels after CPAP treatment (56). Patients with OSA have increased left ventricular mass (measured using echocardiography) compared with non-OSA patients with similar daytime blood pressure values (57). Pulmonary hypertension is not uncommon in OSA (58). These observations in OSA have implications in analysis of data linking obesity and cardiac disease.

The advent of nasal CPAP has prevented large studies investigating the natural history of untreated OSA. He et al. (59) observed an increased cumulative mortality in untreated patients with an apnoea index (AI) > 20 compared to AI < 20. Tracheostomy or CPAP treatment but not uvulopalatopharyngoplasty (UPPP) reduced the mortality risk. A number of groups have reported an increased risk of myocardial infarction and stroke in sleep apnoea (60,61) and untreated OSA may be associated with an increased cardiovascular mortality in patients with coronary artery disease (62). Snoring is a strong risk factor for sleep-related strokes while sleep apnoea symptoms (snoring plus reported apnoeas or EDS) increase the risk of cerebral infarction (odds ratio of 8.0).

**Endocrine Effects**

Sleep apnoea patients are characterized by a neuroendocrine defect in growth hormone (GH) and testosterone secretion (63–66) that may be reversed by nasal CPAP treatment, without associated weight change. It is likely that GH impairment in sleep apnoea is additive to the low GH levels seen in obesity. In adults, impaired GH secretion leads to central adiposity and reduced muscle and bone mass. It is unknown whether these changes in GH and testosterone levels in adults with sleep apnoea are associated with measurable changes in body composition, body fat distribution, energy expenditure or bone density. Recently two reports have suggested that insulin levels are increased in patients with sleep apnoea independent of obesity (67) or visceral fat mass (22). Other data strongly suggest that reversal of sleep apnoea leads to increased insulin sensitivity in obese patients with type 2 (non-insulin-dependent) diabetes (68) and reduced visceral fat mass (69). More recently, leptin has been implicated in sleep-disordered breathing. In obese, leptin-deficient mice with OHS, leptin replacement increased both waking and sleeping minute ventilation and chemosensitivity to carbon dioxide during sleep (70). Leptin levels fell significantly in a group of 22 patients with OSA after 4 days of treatment with CPAP (69), possibly due to reduced sympathetic activity. OSA may well be a confounder in some of the hormonal associations observed in central obesity.
SLEEP-DISORDERED BREATHING—EPIDEMIOLOGY

Recognition of Sleep-disordered Breathing

Clinical impression has surprisingly poor specificity and sensitivity for detecting sleep apnoea (71). Symptoms such as sleepiness are common in the general community including obese patients (72) and may be secondary to lack of sleep, medications or other sleep disorders. Snoring may be underestimated. Physical examination generally has poor predictive value though obvious pharyngeal crowding and tonsillar hypertrophy suggest upper airway obstruction (73).

Breathing during sleep in obese children has attracted much less attention than in the adult population and no prevalence studies have been performed. It appears that obesity is not as dominant a factor in childhood apnoea as it is in adults (74,75).

Prevalence of Sleep-disordered Breathing—General

Results from the Wisconsin Sleep Cohort Study (76) indicate that 9% of female and 24% of male middle-aged public servants have an apnoea index > 5/hour. Using a cut-off of 15 apnoeas per hour (a criterion which would satisfy most sleep researchers), 4% of women and 9% of men have sleep apnoea. Our group has found a similar prevalence of OSA in an Australian rural community using home monitoring of breathing (77).

Sleep Apnoea and Obesity—Epidemiology

All epidemiological investigations have consistently shown that obesity, especially central obesity, is strongly associated with adult sleep-disordered breathing (50,76,77). Measurements of central obesity such as waist or neck measurements are tightly linked to OSA in sleep clinic populations (50). In the Busselton Sleep Survey (77), there was a powerful effect of BMI in increasing the risk of sleep-disordered breathing in the community (Figure 27.2).

There are limited data on the prevalence of sleep apnoea in the obese population. Data from the SOS Study, which examined 3034 subjects with BMI > 35, found that over 50% of obese men and one-third of obese women reported habitual loud snoring (22). In the SOS Study, a history of frequent witnessed apnoeas (a sensitive marker of sleep apnoea in epidemiological studies), was reported by 33% of men and 12% of women. The exact prevalence of the spectrum of sleep-breathing disorders in the obese is unknown but it is clear that OSA and related conditions occur in a very high proportion of obese subjects.

Other Risk Factors for Sleep Apnoea

OSA increases in prevalence with age and is commonly recognized in the 5th to 7th decades. Some of the increase in prevalence with age is due to increased central fat deposition with age.

The male to female ratio in sleep apnoea is close to 2.5:1 (76,77). Sleep apnoea is rare in premenopausal women unless there is morbid obesity (78) or maxillo-facial abnormalities. The prevalence of OSA increases in women after the menopause, leading to speculation that female sex hormones are protective or male hormones may promote OSA. Alternatively, the increased prevalence in OSA after menopause may be secondary to changing body fat distribution in postmenopausal women.

Sleep apnoea aggregates in families and the risk
of having OSA increases progressively with increasing numbers of affected relatives (71). Such risk may be the result of similarities in facial structure affecting upper airway dynamics in sleep. Certain maxillo-facial appearances are linked with sleep apnoea. In obese patients, familial maxillo-mandibular structure will interact to increase the likelihood of sleep apnoea (79). This may explain why weight loss may not be enough to cure sleep apnoea in obese patients (80).

Apart from obesity, conditions causing narrowing of the upper airway will promote the development of sleep apnoea. These include fixed upper airway lesions (e.g. nasal obstruction, enlarged tonsils), macroglossia or neurological conditions impairing upper airway muscle tone (66).

Acute alcohol ingestion promotes apnoea development during later sleep. Lifetime alcohol consumption may be a risk factor for the development of OSA (81). Data from the Wisconsin Sleep Cohort suggests that smoking history may be a dose-dependent risk factor for OSA (82).

A number of endocrine and metabolic disorders apart from obesity are associated with an increased prevalence of OSA. Hypothyroidism may lead to sleep apnoea by reducing chemosensitivity, myxoedematous infiltration of the upper airway and upper airway myopathy (83). Over 50% of patients with acromegaly have sleep apnoea (84). Cushing’s disease is also associated with sleep apnoea.

Cardiac failure (whatever the cause) is associated with a high incidence of sleep-disordered breathing. In a recent study of 450 patients with cardiac failure referred to a sleep laboratory (either with sleep symptoms or persistent dyspnoea), 72% had more than 10 apnoeas–hypopnoeas per hour (85). Patients had OSA or central sleep apnoea (Cheyney–Stokes respiration), with OSA more common in those patients with BMI > 35.

TREATMENT OF SLEEP APNOEA AND SNORING

The approach to treatment will vary according to severity of symptoms, severity of hypoxaemia during sleep and cost. In the absence of significant data showing a deleterious effect of asymptomatic sleep apnoea, treatment for prognosis alone is probably inappropriate. However, patient denial may produce an ‘asymptomatic’ patient, so if there is a highly positive diagnostic study in an ‘asymptomatic’ patient, it is advisable to check with relatives about any symptoms.

Weight Loss

A number of studies have demonstrated a reduction in sleep apnoea severity after weight loss, either through caloric restriction or bariatric surgery. However, it is important to reassess patients after weight loss and ensure there is little residual disordered breathing. Most published reports indicate that, although there is a reduction in apnoea index, a significant degree of apnoea persists, which in most cases warrants further treatment (86–88). Weight loss associated with apparently successful bariatric surgery may have limited efficacy in reducing sleep apnoea as many patients also have maxillo-facial abnormalities predisposing them to OSA (80). Recent data from the SOS Study show a marked reduction in sleep apnoea symptomatology in obese subjects 2 years after surgically induced weight loss compared with controls.

Lifestyle Factors

Some studies have suggested that reduction of smoking and alcohol consumption will lead to reduced self-reported snoring and reverse mild sleep apnoea (89). Sleep deprivation may reduce upper airway tone and chemosensitivity and should be avoided. Drugs such as benzodiazepines or opiates should be avoided at bedtime, particularly in patients with severe OSA or OHS.

Devices

Positive Airway Pressure

Until the early 1980s, tracheostomy was the only form of treatment available for sleep apnoea and was usually performed on patients with severe symptomatic disease. The advent of nasal CPAP revolutionized the management of OSA and allowed a wider range of patients to be treated (90,91). A CPAP machine delivers varying pressure to the
upper airway through a nose or face mask, providing a ‘pneumatic’ splint which prevents upper airway closure. CPAP treatment leads to normalization of sleep architecture, decreased upper airway oedema and a reduction in daytime sleepiness (91,92). CPAP improves cognitive function and quality of life, as well as the associated symptoms listed in Table 27.1 for patients with all degrees of severity of OSA (93). CPAP is not a cure for sleep apnoea. Cessation of treatment will lead to a recurrence of sleep-disordered breathing and accompanying symptoms.

Nasal CPAP is an effective treatment, but compliance is variable (94,95), ranging between 40 and 80%. Problems affecting compliance with nasal CPAP include a sense of claustrophobia, mask air leaks, nasal congestion and dryness of the mouth and throat (usually associated with mask or mouth air leaks), and the inconvenience of using a machine. Patients with mild disease or those requiring high pressures are most likely to be non-compliant. Obese patients generally require higher CPAP pressures (96).

Devices that allow variation between the set inspiratory and expiratory pressures, known as bi-level positive airway pressure were originally introduced to improve compliance in CPAP users (97). Although not proven to improve compliance, this form of positive airway pressure therapy has been used increasingly in the management of severe respiratory failure and hypoventilation during sleep, such as OHS.

**Mandibular Advancement Splints**

The use of an orthodontic device designed to advance the mandible and thus increase the upper airway aperture has produced a major reduction in sleep apnoea severity in several studies (98) and is the subject of a large randomized clinical trial at present in Canada (A. Lowe, personal communication). The efficacy of these devices is likely to be reduced in the obese patient, as skeletal factors are less important in the genesis of upper airway obstruction. In general, these devices are less effective in patients with severe OSA (99). Data on compliance and the prevalence of side effects related to the temporomandibular joint are needed.

**Surgery**

**Tracheostomy**

Prior to the introduction of nasal CPAP as a treatment for OSA, tracheostomy was the major therapeutic modality. Tracheostomy is only currently indicated in patients with severe OSA who have been unable to comply with CPAP or related therapies. Tracheostomy can produce significant morbidity, particularly in the obese fat-necked individual and will be only partly effective in treating OHS. However with skilful minimalist surgery and close follow-up, tracheostomy may be a ‘last-resort’ therapeutic option in some patients.

**Uvulopalatopharyngoplasty (UPPP) and Other Upper Airway Surgery**

This operation was developed for the treatment of heavy snoring in the early 1950s and involves careful removal of the uvula and part of the soft palate. The introduction of UPPP for the treatment of OSA into North America occurred in 1981 (100) but despite early enthusiasm the operation has never lived up to its promise as a ‘cure’ for sleep apnoea (101). There are no preoperative tests that satisfactorily predict the response to surgery. There is a significant morbidity and even mortality (101). Excessive removal of palatal tissue will lead to velopharyngeal incompetence and nasal regurgitation and speech changes. Many studies report particularly poor results in obese patients. More recently UPPP has been performed with a surgical laser or high-frequency radio-waves (‘somnoplasty’), aiming at stiffening palatal tissue rather than complete removal. Meaningful outcome data are lacking and, as in conventional UPPP, subjective reports of snoring improvement are not supported by objective benefit. There is clearly a ‘placebo’ effect in snoring surgery that has been demonstrated in other forms of surgical intervention, such as simple sternotomy for severe angina.

More complex maxillo-facial surgery, usually involving UPPP in combination with genioglossus advancement via a mandibular osteotomy and hyoid myotomy, has been used with some success in the treatment of OSA. However, this surgery is less effective in patients with severe disease ( > 60 events per hour and desaturation to 70%) and in the morbidly obese (102).
Many centres prefer to manage these patients in hospital, even for brief periods. While most patients starting CPAP require only one night of sleep monitoring to adequately determine required pressure, patients with sleep apnoea and awake respiratory failure require more detailed assessment. In these patients, oxygen alone should be used with caution and with close monitoring of hypercapnia. This is one group in whom sedation or use of hypnotics is contraindicated. Until recently, high CPAP pressures or CPAP plus added oxygen were needed in the first weeks of treatment until blood gases improved (91,103) or, for the obtunded hypercapnic patient, a short period of intubation and ventilation may have been required. Currently, the bi-level positive airway pressure systems can deliver effective non-invasive pressure support ventilation to these patients and successfully treat hypercapnic respiratory failure (Figure 27.3). Home use of these devices is then prescribed with or without oxygen, depending on the degree of intrinsic lung disease.

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